Endovascular repair of aortic allograft aneurysmal degeneration: A case report

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Aortoenteric graft fistula remains a dreadful complication of aortic surgery. Good results have been reported using in situ graft replacement with arterial allografts. Late aneurysmal degeneration of the graft itself may necessitate further repair. We report the case of such an aneurysmal degeneration 7 years after implantation of the allograft. Endovascular repair was performed with a Vanguard device; complete exclusion was obtained immediately. At 6-month follow-up, the patient was alive and well. Duplex and computed tomography scans showed an excluded aneurysm with a slight reduction in size. Endovascular stent grafting may be a therapeutic option for treating patients with late allograft degeneration. (J Vasc Surg 2000;32:1222-4.)

Treatment of aortic graft infection remains a surgical challenge. In 1993, Kieffer et al[1] reported their results of 43 patients in whom they performed in situ replacement with arterial allografts. This method is now widely used in our country. However, degeneration and dilatation of arterial allografts are well-known late complications requiring close follow-up of these patients.[2] Until recently, surgical replacement with a new graft was the only available treatment of such dilatations. We report the endovascular repair of an aneurysmal allograft degeneration by means of a bifurcated stent graft.

CASE REPORT

Progressive enlargement of an arterial allograft was detected in a 65-year-old man during follow-up. In 1981, this patient underwent an aortobifemoral bypass grafting for intermittent claudication. The graft was made of woven Dacron. In 1991, infection of the graft was detected, and aortic graft–enteric fistula was discovered during surgery. Bacterial contamination of the graft was massive with several microorganisms cultured: *Enterococcus* species, *Staphylococcus aureus*, and *Candida albicans*. Surgical treatment consisted of the removal of all prosthetic material, suturing of the duodenum, in situ replacement with a fresh arterial allograft, and prolonged antibiotherapy. The arterial allograft was a fresh graft harvested during a multiorgan procedure. This was a composite graft: the proximal portion was a segment of descending thoracic aorta, the distal portion was an arterial segment including the distal part of the abdominal aorta and both iliac arteries, and both segments were anastomosed end-to-end before implantation. The allograft was fixed 2 cm below the renal arteries in an end-to-end fashion. Postoperative course was marked by bilateral hydronephrosis treated with ureteral catheters. At 3 months postoperatively, intestinal occlusion necessitated surgical open repair. From this point on, late follow-up was uneventful, the patient did not have any claudication, and regular computed tomography (CT) scans were obtained to detect any enlargement of the allograft. This enlargement appeared progressively. The initial diameter of the body of the graft was 28 mm, and after the first year, annual growth of the diameter was 3 mm, reaching 5 mm the last year before the procedure. At 7-year follow-up, the proximal part of the allograft became truly aneurysmal with a diameter of 48 mm. On examination, the patient did not complain of pain on abdominal palpation. All peripheral pulses were present, and the patient did not have any claudication. The results of blood tests were in the normal range without any inflammatory syndrome, and C-reactive protein was normal. We did not use any other test to rule out recurrent infection of the allograft because most likely, late degeneration of the graft was the cause of the dilatation.

An aorto-arteriography was performed with a marked catheter, showing a dilatation of the body of the allograft and the presence of a long neck (> 4 cm) above the aneurysm (Fig 1). The branches of the allograft were patent with a good runoff. Occlusion of the inferior mesenteric artery and both internal iliac arteries was noted. On the CT, the aneurysmal dilatation was limited to the body of the allograft, and mural thrombus was present in the aneurysmal sac. Endovascular treatment was proposed to this patient. A bifurcated stent graft was placed through a right groin incision and a left percutaneous puncture of the femoral artery. We used the Vanguard endoprosthesis

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Fig 1. Preoperative aortography showing a long proximal neck above the aneurysmal degeneration of the allograft.

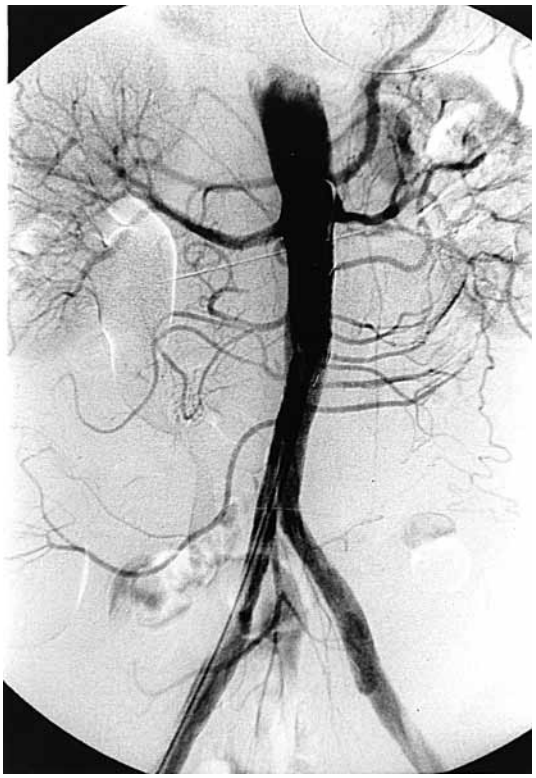

Fig 2. Final angiography showing complete exclusion of the aneurysm.

(Boston Scientific Corporation, Boston, Mass). This is a self-expandable device with a thermic form memory. The main body of the device was placed through the right femoral artery. Its proximal diameter was 22 mm, the distal diameter was 10 mm, and the length was 153 mm. The proximal endograft attachment site was just below the renal arteries in the native aorta, completely covering the proximal part of the allograft. The contralateral limb was introduced percutaneously through a 10-F sheath placed in the left femoral artery. The limb was 100 mm long and had a diameter of 10 mm. Gentle manual inflation was carried out with a 10/4 balloon. Retrograde control angiography showed bilateral distal endoleaks that were treated with extensions of increasing diameters (12-14 mm) in both iliac limbs of the allograft. Final control showed complete exclusion of the aneurysmal dilatation of the allograft without any visible leak (Fig 2). The procedure time was 130 minutes, with 35 minutes of fluoroscopy and a 170-mL injection of contrast medium. After the procedure, recovery was uneventful. The patient was discharged on the fourth day after the procedure, and a control CT scan with contrast injection showed perfect patency of the device without any apparent leak. At 6-month follow-up, the patient was asymptomatic. Duplex scan showed good patency of the device without any leak, and a new control CT showed perfect exclusion of the aneurysmal sac with a slight reduction of the aneurysmal diameter (45 mm) (Fig 3).

DISCUSSION

Treatment of aortoenteric graft fistula remains a surgical challenge with early mortality ranging from 19% to 38%.[3,4] Recurrent infection and limb loss occur in a substantial number of patients. Two different therapeutic approaches are currently used: first, complete resection of the infected graft, associated with surgical closure of the aorta and extra-anatomic bypass grafting, and second, in situ reconstruction after removal of the infected prosthesis, with the use of either a new prosthetic graft or arterial or venous allografts. Since 1990,[5] we have treated 14 patients with an infected infrarenal aortic prosthetic graft; six of them had an aortic graft–enteric fistula. Prolonged and extensive follow-up of these patients was maintained to detect any late deterioration of the allografts.[2,6] This deterioration can occur after two modes: stenosis and dilatation. The first is mainly due to intimal hyperplasia in the branches of the graft leading to progressive occlusion. Its incidence can reach 20%,[4] and it can be treated with either ballon angioplasty or reoperation. The dilatation of the graft seems to be less frequent and could be due to the use of the descending thoracic aortic allografts.[4] In our patient, progressive enlargement

Fig 3. Control CT scan at 6 months showing graft's limb patency and complete exclusion of the aneurysm.

of the body of the graft led us to an aggressive approach regarding the aneurysmal allograft. Surgical reoperation would have been the "classic" mode of treatment for this patient, with in situ replacement of the body of the allograft with a new prosthetic graft in an aseptic field. Such reoperation is often challenging because of dense adhesions and hemorrhagic risk. An endovascular approach was discussed to reduce the surgical morbidity. Since the original work by Parodi et al,[7] many publications have stressed the feasibility of endovascular treatment of aortic aneurysms.[8-10] Recent reports have emphasized the incidence of early and late complications, the most frequent being endoaneurysmal endoleaks. Late rupture has also been reported without any evidence of endoleak.[11] Despite these problems, endovascular treatment of aortic aneurysm remains an attractive method, particularly for elderly patients with a high surgical risk. We have treated 50 patients using either the Vanguard or the Talent device. The present case was particularly suitable for endovascular treatment because of the following reasons: the infrarenal neck was long (> 4 cm), the iliac branches of the allograft were linear, and the internal iliac arteries were occluded. Furthermore, this case compares favorably with other postaortic reconstruction aneurysmal complications such as anastomotic pseudoaneurysms. Because the aneurysmal degeneration was restricted to the body of the allograft, we could treat a straight fusiform sac without particular caution or difficulty because of the renal or internal iliac arteries. The placement of the endovascular device was then particularly easy, and immediate exclusion of the aneurysm was obtained. Care was taken to overlap the anastomosis between the allograft and the native aorta to allow placement of the proximal attachment site in the native aorta and avoid any potential late complications such as a pseudoaneurysm between the aorta and the allograft. The main concerns about endovascular treatment of allograft aneurysmal dilatation are related to the future of the allograft itself. First, the part of the allograft covered by the endovascular device could become ischemic because of the radial forces applied by the device. Second, the remaining part of the graft (ie, the branches) could also degenerate mainly because of progressive stenosis or occlusion. To our knowledge, the interactions between an endovascular device and an allograft are not known. We did observe medial alterations (necrosis) in a sheep model of aortic endovascular grafting with oversizing of the device (M. Matem, unpublished data, Sep 1996). Furthermore, late stenosis of the branches of arterial allografts may occur. We do not know yet if the presence of the endovascular device inside these branches will cause further hyperplasia, particularly below the distal attachment site. Aggressive follow-up is obviously needed including CT and duplex scanning.

In summary, to our knowledge this is the first case of late aneurysmal degeneration of an arterial allograft treated with an endovascular device. This method could become the treatment of choice for such late degenerations, avoiding challenging surgical reoperations.

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